



Treatment of Ventricular Tachycardia Using an Automatic Scanning Extrastimulus Pacemaker

C PRATAP REDDY, MD, FACC, EDWARD P TODD, MD, FACC, CHIEN S KUO, MD, FACC, ANTHONY N DeMARIA, MD, FACC

Lexington, Kentucky

A patient with recurrent sustained ventricular tachycardia that was resistant to both conventional and experimental antiarrhythmic agents was treated with a programmable automatic scanning extrastimulus pacemaker. The antitachycardia pacemaker was implanted only after many episodes of spontaneous and laboratory-induced ventricular tachycardia were reliably and reproducibly terminated with programmed ventricular extrastimuli. In the 6 months since implantation of the

automatic scanning pacemaker, all episodes of ventricular tachycardia have been terminated successfully by the pacemaker. Acceleration of rate of ventricular tachycardia or induction of ventricular fibrillation did not occur at any time during attempted termination of ventricular tachycardia by the pacemaker.

The advantages of the automatic scanning extrastimulus pacemaker over other antitachycardia pacemakers are discussed.

Recognition that many cases of recurrent sustained ventricular tachycardia can be reproducibly terminated by programmed ventricular stimulation has led to the use of many pacing techniques in the long-term management of recurrent ventricular tachycardia (1-7). These techniques include underdrive and burst overdrive pacing, and externally-activated programmed extrastimulation (1-7). However, no one of these modalities has received wide application because of the technical limitations inherent in the available pacemakers and the potential risk of acceleration of ventricular tachycardia and induction of ventricular fibrillation by some of the currently used pacing techniques (3,8,9). Recently, a programmable automatic scanning extrastimulus pacemaker that overcomes many of these technical and practical problems has been introduced and successfully used in the treatment of paroxysmal supraventricular tachycardia (10). To our knowledge, the use of this pacemaker for treatment of ventricular tachycardia has not been reported previously. In this report, we describe the use in one patient of such a

pacing system for long-term management of recurrent ventricular tachycardia

Case Report

A 64 year old man was transferred to the University of Kentucky Medical Center from a regional hospital on September 7, 1982 for electrophysiologic study and management of recurrent sustained ventricular tachycardia. In April 1982, the patient had an inferior wall myocardial infarction from which he recovered uneventfully. After the infarction, he remained asymptomatic except for occasional exertional angina pectoris. Forty-three days before his transfer to this center, the patient was admitted to a regional hospital because of palpitation and chest pain. An electrocardiogram recorded at that time showed ventricular tachycardia which was terminated by direct current cardioversion (Fig 1). During his stay in the hospital he continued to have recurrent episodes of ventricular tachycardia requiring direct current cardioversion. Therapeutic plasma concentrations of conventional antiarrhythmic drugs, including lidocaine, procainamide, quinidine, phenytoin and propranolol, either alone or in various combinations, failed to prevent the recurrence of ventricular tachycardia.

Physical findings. Physical examination revealed a mildly obese man in no acute distress. The blood pressure was 150/90 mm Hg, irregular pulse 60/min and respirations 16/min. The carotid pulses were normal. Examination of

From the Cardiovascular Division, Department of Medicine and the Department of Cardio-Thoracic Surgery, Albert B. Chandler Medical Center and the University of Kentucky Medical School, Lexington, Kentucky. Manuscript received April 4, 1983, revised manuscript received July 18, 1983, accepted July 22, 1983.

Address for reprints: C. Pratap Reddy, MD, Cardiovascular Division, Department of Medicine, University of Kentucky Medical School, Lexington, Kentucky 40536.

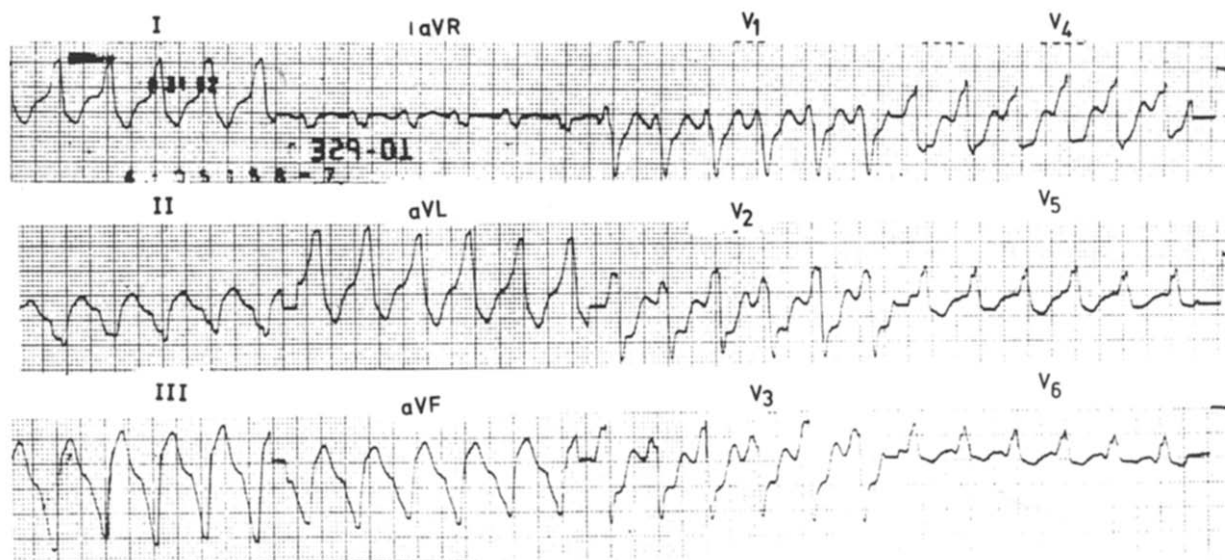


Figure 1. Twelve lead electrocardiogram recorded during ventricular tachycardia.

the heart revealed normal first and second heart sounds. There was a grade 2/6 systolic ejection murmur in the aortic area. The lungs were clear. The abdomen and limbs were normal.

Laboratory data. The electrocardiogram revealed sinus rhythm, first degree atrioventricular block and old inferior wall myocardial infarction. The chest X-ray film showed mild cardiomegaly. Blood chemistry values, including urea nitrogen, creatinine and serum electrolytes, were within normal limits. Echocardiography revealed an enlarged left atrium, borderline left ventricular hypertrophy, thickened aortic valve leaflets and inferoposterior and lateral wall hypokinesia.

Cardiac catheterization revealed total occlusion of the distal circumflex coronary artery, 25% obstruction of the right coronary artery and a normal left anterior descending coronary artery. The left ventricular angiogram revealed a poorly contracting ventricle with akinesia of the inferoposterior wall.

Electrophysiologic study. Electrophysiologic study for induction of ventricular tachycardia and short-term drug testing was done using the standard techniques for His bundle recordings and programmed ventricular stimulation (5,8). Ventricular tachycardia could be reproducibly induced and terminated by programmed ventricular extrastimuli or by rapid ventricular pacing (Fig. 2). To test the efficacy of high dose procainamide (5) in preventing initiation of tachycardia, 1 g of procainamide was infused intravenously and programmed stimulation repeated. After procainamide infusion, ventricular tachycardia could still be induced. Because of failure of high dose procainamide to prevent initiation of tachycardia and the clinically proven failure of

other antiarrhythmic agents, treatment with investigational antiarrhythmic agents was recommended (11).

Hospital course and treatment. The patient was admitted to the coronary care unit and treatment was begun with amiodarone, 1,200 mg/day. After 8 days of treatment, ventricular tachycardia continued to occur (though less frequently and at a slower rate) and some of the episodes required programmed ventricular stimulation or direct current shock for termination. Because of continued occurrence of tachycardia, amiodarone was discontinued and the patient was treated with mexiletine, mexiletine and quinidine combined, and aprindine in that order over a 15 day period. Mexiletine alone or in combination with quinidine failed to suppress tachycardia and aprindine aggravated the arrhythmia and caused as many as 30 attacks of tachycardia a day.

Pacemaker implantation. Because of failure of conventional and investigational antiarrhythmic agents to prevent recurrence of tachycardia, the patient was advised to undergo antiarrhythmic cardiac surgery or have an anti-tachycardia pacemaker implanted. The patient elected to have a pacemaker implanted. It was believed that in the absence of a discrete ventricular aneurysm that could be resected, implantation of an antitachycardia pacemaker was probably the most appropriate choice. In view of reproducible termination of tachycardia by programmed stimulation in both the electrophysiology laboratory and the coronary care unit, implantation of a programmable automatic scanning pacemaker (Teletronics model 4151 "PASAR") was recommended.

To determine the most appropriate coupling intervals to be programmed into the pacemaker for termination of tachycardia, a repeat electrophysiologic study was performed. Ventricular tachycardia was induced and the tachycardia termination window was determined by scanning the diastole with double ventricular extrastimuli (S_1 and S_2) at

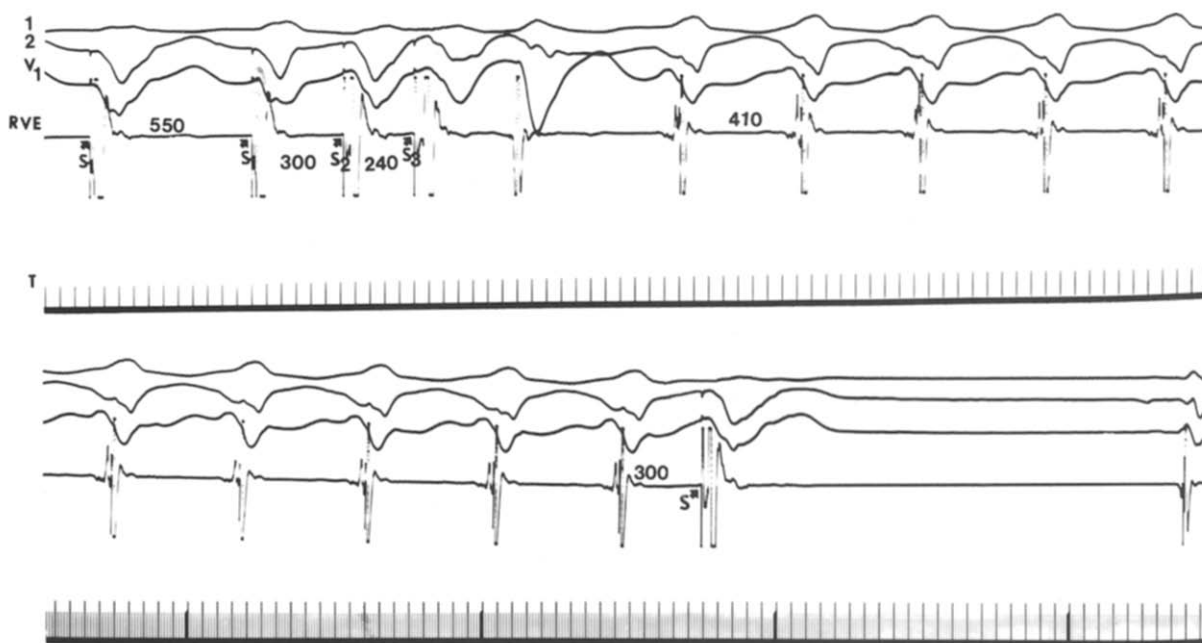


Figure 2. Initiation and termination of ventricular tachycardia by programmed ventricular extrastimuli. The tracings are organized in each panel from top to bottom as follows: Electrocardiographic leads I, II and V₁ and electrogram from right ventricular apex (RVE) and time (T) lines at 10 and 50 ms intervals. Initiation of ventricular tachycardia by double extrastimuli (S₂ and S₃) coupled to the basic drive beat (S₁) is shown in the **top panel**. Termination of tachycardia by a single extrastimulus (S₄) is shown in the **bottom panel**.

decremental intervals of 10 ms (Fig 3). During scanning, the coupling interval of S₁ was decreased and the interval between S₁ and S₂ remained fixed (Fig 3). The appropriate coupling intervals for S₁ and S₂ extrastimuli were then programmed into the pulse generator and the pacemaker was implanted using conventional techniques on the 29th day of hospitalization. Immediately after implantation of the pacemaker, several episodes of both induced and spontaneous tachycardia were successfully terminated by the pacemaker (Fig 4). During his hospital stay, the patient had several episodes of tachycardia, all of which were promptly terminated by the pacemaker. Two days after implantation of the antitachycardia pacemaker, the patient was restarted on amiodarone therapy because of its beneficial effects on the tachycardia noted earlier.

Description of pacemaker. The Teletronics model 4151 ("PASAR") is a bipolar lithium-powered pacemaker that can be programmed transcutaneously after implantation. The pacemaker automatically becomes activated when four consecutive cycles of tachycardia have occurred at a rate faster than a preprogrammed tachycardia detection rate. After the fourth complex of tachycardia, a single extrastimulus (S₁) or double extrastimuli (S₁ and S₂) (depending on program-

ming) are discharged at preset coupling intervals from the sensed tachycardia QRS complex. If tachycardia is not terminated, the pacemaker discharges a further stimulus (or stimuli) after every four cycles and for each stimulus the coupling interval is reduced by 6 ms until tachycardia is terminated or 90 ms of diastole have been scanned. When two extrastimuli are programmed (as in the case reported here), the coupling interval between the two extrastimuli (S₁ and S₂) stays fixed while the interval between the tachycardia complex and the S₁ decreases during the scan. If tachycardia has not terminated after a 90 ms scan, the pacemaker recycles and the scan is repeated.

When tachycardia is terminated, the pacemaker ceases to discharge extrastimuli and the timing of the extrastimuli that successfully terminated the tachycardia is retained in the pacemaker memory, at the onset of the next episode of tachycardia, the pacemaker starts the scan using the previously successful timing variables kept in its memory.

Follow-up. The patient has been followed up closely since his discharge from the hospital. He has remained asymptomatic, has had no clinical evidence of sustained tachycardia and has not required hospitalization. Ambulatory electrocardiographic monitoring studies have revealed at least 16 episodes of tachycardia during which the pacemaker functioned as expected and successfully terminated all episodes of tachycardia within an average of 8 seconds (range 3 to 25) of onset of tachycardia (Fig 5). During the recorded episodes of tachycardia, the patient was asymptomatic. In the 6 months since his discharge from the hospital, the combination of the antitachycardia pacemaker and amiodarone therapy has provided safe and effective control of ventricular tachycardia. Because of changes in the rate of ventricular tachycardia and refractoriness of ventricular

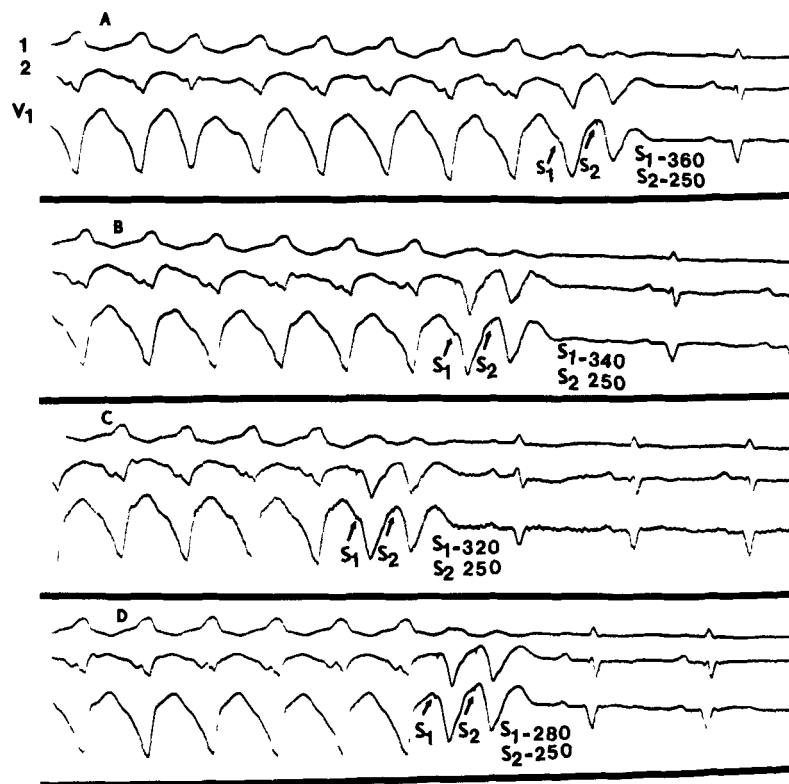


Figure 3. Determination of tachycardia termination window. In each panel from top to bottom are electrocardiographic leads I, II and V₁. Termination of induced ventricular tachycardia is produced by double ventricular extrastimuli (S₁ and S₂) delivered at different coupling intervals. Note that while the interval between the last tachycardia complex and S₁ is shortened, the interval between S₁ and S₂ stays fixed. The range of coupling intervals from 360 to 260 (coupling interval of 260 not shown in the figure) defines the tachycardia termination window.

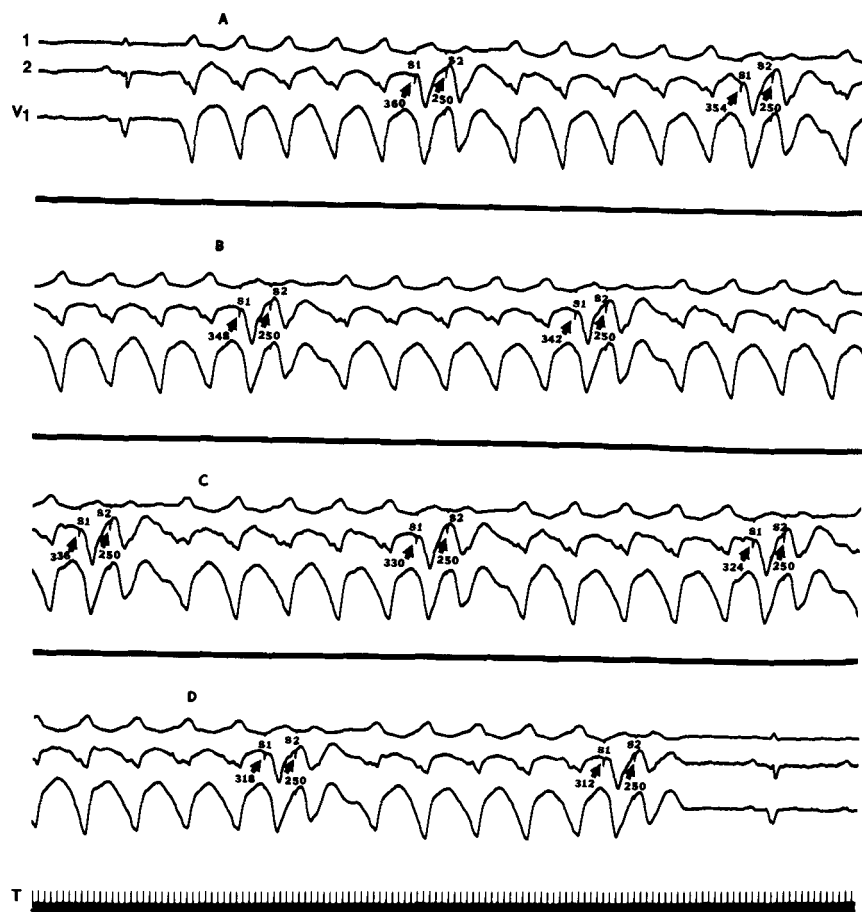


Figure 4. Termination of spontaneously occurring tachycardia by the implanted pacemaker. Tracings are arranged as in Figure 3. Note that the pacemaker is activated as soon as four tachycardia cycles have occurred at a rate faster than the programmed pacemaker trigger rate of 140 beats/min (A). Pacemaker scanning continues until the tachycardia is terminated (D). For each stimulus the coupling interval of S₁ is shortened by 6 ms while the interval between S₁ and S₂ stays fixed. Tachycardia was terminated in 24 seconds. T = time lines.

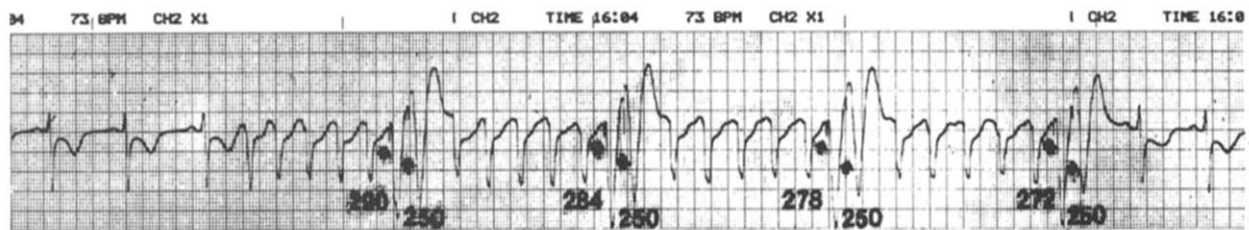


Figure 5. Ambulatory electrocardiographic monitoring showing the onset of tachycardia and its termination by the pacemaker. The **arrows** indicate the extrastimuli discharged by the pacemaker and the **numbers** indicate the coupling intervals of extrastimuli bpm = beats per minute

myocardium produced by amiodarone, S_1 and S_2 were moved to longer coupling intervals twice (5 and 24 weeks, respectively, after initiation of amiodarone treatment) since the patient's discharge from the hospital. The effectiveness of the newly programmed intervals was confirmed each time by induction of tachycardia using the same pacemaker.

Although the memory feature of the pacemaker was useful, the "remembered" S_1 and S_2 intervals were ineffective in terminating tachycardia in 30% of recorded episodes. However, each of these episodes was terminated by premature stimuli located within the 90 ms search zone.

Discussion

Available evidence suggests that most sustained ventricular tachycardias are due to a reentrant mechanism (12,13). Programmed stimulation studies (1,8,9,12,13) have shown that reentrant ventricular tachycardias may be terminated by critically timed single or double ventricular extrastimuli or rapid ventricular pacing. Termination of ventricular tachycardia by these techniques is presumably due to interruption of reentry by paced stimuli. A single premature stimulus may fail to terminate the tachycardia because it is unable to reach or penetrate the site of origin of tachycardia. However, the same tachycardia may be successfully terminated by double premature stimuli because shortening the refractory period of the ventricle by the first premature beat allows the second premature beat to penetrate and interrupt the reentrant circuit (9,13). The same mechanism is invoked to explain the interruption of tachycardia by rapid pacing.

Previous experience with pacemaker management of ventricular tachycardia. *Burst pacing.* To date, burst ventricular pacing (manually activated, automatic or radiofrequency) has been the most commonly used pacing modality in the long-term management of ventricular tachycardia. Fisher et al (2) used burst pacing in 13 cases of ventricular tachycardia (6 radiofrequency, 4 automatic and 3 manual with magnet) and reported good long-term results.

During a mean follow-up period of 18 months, 2 of 13 patients required emergency admission for control of ventricular tachycardia that was not terminated by pacing and 3 patients died suddenly. However, none of the deaths were attributed to pacemaker malfunction. Hartzler et al (4) and Ruskin et al (6), who used patient-activated radiofrequency ventricular pacing with or without concomitant drug therapy for control of ventricular tachycardia, reported similar results. Despite favorable results of pacing in these small series of patients, concern for possible acceleration of the rate of ventricular tachycardia or the development of pacing-induced ventricular fibrillation has limited a more widespread use of burst pacing in the management of ventricular tachycardia. Fisher et al (3) reported a 4% incidence rate of burst pacing-induced acceleration of ventricular tachycardia and a 1% incidence rate of pacing-induced ventricular fibrillation during attempted termination of 573 episodes of ventricular tachycardia in 23 patients. However, tachycardia acceleration occurred at least once in 43% of the 23 patients.

Programmed ventricular extrastimuli. In contrast to burst pacing, programmed ventricular extrastimuli often successfully terminate ventricular tachycardia, and the potential risk of speeding the rate of tachycardia and induction of ventricular fibrillation may be low (8,12-14). In the series of Wellens et al (12) and Josephson et al (13), ventricular tachycardia was terminated by single or double premature stimuli without causing acceleration of ventricular tachycardia or induction of ventricular fibrillation. However, in the series of Spielman et al (14), ventricular fibrillation occurred in 8% of 125 patients studied with double premature ventricular stimuli. Despite the high success rate of programmed extrastimuli in terminating ventricular tachycardia, pacemakers capable of delivering timed extrastimuli have not been developed and used until recently.

In the long-term management of patients with ventricular tachycardia, Kappenberger and Sowton (15) and Greene et al (16) successfully used an externally-activated permanent pacemaker capable of delivering timed extrastimuli. However, this system is not versatile because it must be manually activated by the patient and the extrastimuli can be delivered only at fixed intervals. The latter feature makes this pacemaker unreliable because the rate of ventricular tachycardia, the tachycardia termination window and the electrophysiologic properties of the ventricles vary in response to changes in autonomic tone, presence or absence of myocardial ischemia and concomitant drug therapy.

Advantages of pacemaker used in this study. The pacing system described in this report has been designed to overcome many of the problems just described. 1) It is fully automatic and does not depend on an external control device or patient cooperation for its activation. These features make this pacemaker suitable for patients who suffer from frequent episodes of tachycardia, are unable to activate the device or are unaware of the tachycardia before it leads to cardiac decompensation. The pacemaker activates automatically after only four beats of tachycardia and thus has the advantage of converting the tachycardia within a few seconds before serious hemodynamic derangement occurs. 2) One or two extrastimuli can be used. 3) The pacemaker can scan diastole to find the correct coupling intervals for the extrastimuli for termination of tachycardia. This feature overcomes the problems associated with pacemakers designed to deliver fixed extrastimuli and can be relied on to terminate the tachycardia even when changes occur in the tachycardia termination window. 4) The pacemaker is unique in that the timing of extrastimuli that successfully terminated the tachycardia is retained in the pacemaker memory, and at the onset of the next episode of tachycardia, the pacemaker starts its scan using the previously successful timing variables kept in memory. However, the remembered sequence was not always effective in terminating the tachycardia in our patient. 5) The implanted pulse generator can be programmed after implantation to permit adjustment of the scanning period, the coupling intervals and the pulse width. These features reduce the risk of pacemaker-induced arrhythmias.

Before an antitachycardia pacemaker is implanted, a detailed electrophysiologic study should be performed to determine the tachycardia termination window and the most appropriate coupling intervals for the extrastimuli and to ensure that the selected extrastimuli terminate each episode of induced ventricular tachycardia without adverse effects.

This pacemaker system represents a useful and versatile form of therapy, either alone or as an adjunct to drug treatment for selected cases of recurrent sustained ventricular tachycardia. However, its safety, reliability and long-term effectiveness are unknown and more experience is needed before definite conclusions can be made. Another limitation of this pacemaker is the absence of a back-up demand pacing feature. Also, it must be emphasized that this approach is not designed to prevent ventricular tachycardia but only to terminate it after its occurrence. Consequently, pacemakers are not a substitute for therapy directed at prevention or cure of tachycardia, but must be a part of an overall comprehensive strategy to combat this life-threatening arrhythmia.

Implications. It is not known how many patients with ventricular tachycardia may benefit from a permanent antitachycardia pacemaker because all patients are initially treated with one or more antiarrhythmic drugs and only

patients refractory to drug treatment are considered for pacemaker therapy. In the series of Fisher et al (2) and others (4,5), only 6 to 8% of patients with ventricular tachycardia received permanent pacemakers for arrhythmia control. However, in light of the inconvenience and side effects associated with long-term drug therapy, an antitachycardia pacemaker probably will become the treatment of choice in more patients when more versatile and reliable pacemakers are available.

References

- Wellens HJJ, Bar FW, Gorgels AP, Muncharaz JF. Electrical management of arrhythmias with emphasis on the tachycardias. *Am J Cardiol* 1978;41:1025-34.
- Fisher JD, Kim SG, Furman S, Matos JA. Role of implantable pacemakers in control of recurrent ventricular tachycardia. *Am J Cardiol* 1982;49:194-206.
- Fisher JD, Mehra R, Furman S. Termination of ventricular tachycardia with bursts of rapid ventricular pacing. *Am J Cardiol* 1978;41:94-102.
- Hartzler GO, Holmes DR Jr, Osborn MJ. Patient-activated transvenous cardiac stimulation for the treatment of supraventricular and ventricular tachycardia. *Am J Cardiol* 1981;47:903-9.
- Josephson ME, Horowitz LN. Electrophysiologic approach to therapy for recurrent sustained ventricular tachycardia. *Am J Cardiol* 1979;43:631-42.
- Ruskin JN, Garan H, Poulin F, Harthorne JW. Permanent radiofrequency ventricular pacing for management of drug-resistant ventricular tachycardia. *Am J Cardiol* 1980;46:317-21.
- Griffin JC, Mason JW, Ross DL, Calfee RV. The treatment of ventricular tachycardia using an automatic tachycardia terminating pacemaker. *PACE* 1981;4:582-8.
- Mason JW, Winkle RA. Electrode-catheter arrhythmia induction in the selection and assessment of antiarrhythmic drug therapy for recurrent ventricular tachycardia. *Circulation* 1978;58:971-85.
- Reddy CP, Sartini JS. Nonclinical polymorphic ventricular tachycardia induced by programmed cardiac stimulation: incidence, mechanisms and clinical significance. *Circulation* 1980;62:988-95.
- Spurrell RAJ, Nathan AW, Bexton RS, Hellestrand KJ, Nappholz T, Camm AJ. Implantable automatic scanning pacemaker for termination of supraventricular tachycardia. *Am J Cardiol* 1982;49:753-60.
- Waxman HL, Buxton AE, Sadowski LM, Josephson ME. The response to procainamide during electrophysiologic study for sustained ventricular tachyarrhythmias predicts the response to other medications. *Circulation* 1983;67:30-7.
- Wellens HJJ, Duren DR, Lie KI. Observations on mechanisms of ventricular tachycardia in man. *Circulation* 1976;54:237-44.
- Josephson ME, Horowitz LN, Farshidi A, Kaster JA. Recurrent sustained ventricular tachycardia. I. Mechanisms. *Circulation* 1978;57:431-40.
- Spielman SR, Farshidi A, Horowitz LN, Josephson ME. Ventricular fibrillation during programmed ventricular stimulation: incidence and clinical implications. *Am J Cardiol* 1978;42:913-8.
- Kappenberger L, Sowton E. Programmed stimulation for long-term treatment and non-invasive investigation of recurrent tachycardia. *Lancet* 1981;1:909-14.
- Greene HL, Gross BW, Preston TA, et al. Termination of ventricular tachycardia by programmed extrastimuli from an externally activated permanent pacemaker. *PACE* 1982;5:434-9.